In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 19-397V

JANE B. RUSHING,	*	Chief Special Master Corcoran
Petitioner,	*	Filed: February 16, 2024
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Nancy R. Meyers, Turning Point Litigation, Greensboro, NC, for Petitioner.

Jennifer A. Shah, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT[1]

On March 14, 2019, Jane B. Rushing filed this action seeking compensation under the National Vaccine Injury Compensation Program (the "Program").[2] Petition (ECF No. 1). Petitioner alleges the Table claim that an influenza ("flu") vaccine she received on November 20, 2012, caused her to incur Guillain-Barré syndrome ("GBS"). *Id.*

Although it would seem a claim based on a vaccine received over six years before the Petition's filing might be untimely, Petitioner relies on the "lookback" provision of the Vaccine Act, which allows a claim based upon an injury newly-added to the Table to be considered timely,

[1] Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public in its present form. *Id.*

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter "Vaccine Act" or "the Act"]. Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

if the claim has been initiated not later than two years after the effective date of the Table revision (and provided the alleged injury itself occurred within eight years before the effective date). *See* Section 16(b)).

Petitioner alleges that she meets the requirements of a flu vaccine-GBS Table claim, and therefore the lookback "saves" the claim—the Table was amended to add such a claim in 2017, and this Petition was filed within the two-year, post-amendment window. Respondent disagrees, maintaining that Petitioner's injury (a) has another explanation (the chemotherapy regime she had undergone pre-vaccination), and (b) is otherwise not properly understood to be GBS. The parties have offered expert reports, treater opinions, and briefs in support of their respective positions. *See* Petitioner's Brief, dated May 9, 2023 (ECF No. 57) ("Br."); Respondent's Opposition, dated July 19, 2023 (ECF No. 60) ("Opp."); Petitioner's Reply, dated Aug. 24, 2023 (ECF No. 61). Now, I find that Petitioner has met the flu vaccine-GBS Table requirements—and therefore the lookback does indeed "save" the claim.

I. Fact History

Pre-Vaccination Conditions

Ms. Rushing's past medical history included a number of chronic conditions, including hypertension, myocardial infarction, coronary artery disease, osteoporosis, and gastroesophageal reflux disease. Ex. 4 at 9466. Certain treatment she had been receiving in the months prior to the relevant vaccination has special relevance herein.

Specifically, on June 14, 2012, Petitioner had a right mastectomy after being diagnosed with breast cancer. Ex. 4 at 9465. In the ensuing two months, she underwent four cycles of chemotherapy. *Id*. Thereafter, from September 13 to December 7, 2012, she received weekly secondary chemotherapy treatments of Taxol,[3] an antineoplastic agent used to treat breast and other cancers. *Id*. In this timeframe, Petitioner received 960 mg/m^2 cumulative dose of the drug. *See* Ex. E at 2 (Dr. Callaghan's supplemental expert report). As discussed in more detail below, Respondent contends that Petitioner's symptoms could be attributed to this chemotherapy regime, although Petitioner does not agree.

The medical record also establishes that Petitioner may have been experiencing neuropathic symptoms well before the subject vaccination (but after her chemotherapy regime had been started). On October 12, 2012, more than one month before the vaccination, Ms. Rushing saw her oncologist, Dr. Garry Schwartz, after her fourth weekly Taxol treatment. Ex. 13 at 24–25.

[3] Taxol is defined as "a microtubule-stabilizing drug that is approved by the Food and Drug Administration for the treatment of ovarian, breast, and lung cancer, as well as Kaposi's sarcoma." B. Weaver, *How Taxol/Paclitaxel Kills Cancer Cells*, 25 Mol Biol Cell 2677 (2014), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4161504/ (last visited Feb. 16, 2024).

At this time, she complained of generalized fatigue, a seven-pound weight loss since her last visit, and numbness that radiated from her armpit down to her right hand. *Id.* Dr. Schwartz could not reproduce the numbness on exam, though he opined that it was mechanical in origin. *Id.* A month later, on November 7, 2012, she saw Dr. Schwartz again and reported that the Taxol treatment was causing other kinds of symptoms (weight loss or a feeling of coldness). Ex. 13 at 26. On exam, she was hypotensive and displayed thickened extremities with non-pitting edema. *Id.*

Vaccination and Neuropathic Concerns

Petitioner was sixty-eight years old when she received the flu vaccine on November 20, 2012. Ex. 1 at 2. The record reveals no immediate vaccine reaction, but ten days later (November 30, 2012) she saw Nurse Tina Evans in Dr. Schwartz's office and reported "continuing neuropathy in her hands" that had "been ongoing for the past 2-3 [weekly] treatments." Ex. 13 at 28. The record indicates that Petitioner was deemed to have "stage III invasive lobular carcinoma of the right breast," and had "completed 4 cycles of dose dense Adriamycin/Cytoxan and has now completed 10 weekly doses of Taxol. She is due for dose 11 today." *Id.* However, on December 3, 2012, Petitioner called Dr. Schwartz's office, noting that although she was that morning experiencing some dizziness and weakness, she overall felt a little better. *Id*. at 37.

On December 7, 2012, Petitioner saw Dr. Schwartz for another appointment (she was scheduled to complete her Taxol treatment at this time). Ex. 3 at 29. The record of this visit includes Dr. Schwartz's comment that Petitioner "overall look[ed] well, but the cumulative effects of chemotherapy seem[ed] to be wearing her down somewhat." *Id*. Petitioner reported mild nausea and minor weight loss since her previous visit, but her main complaints at that time were myalgias and arthralgias, particularly of the lower extremities, plus some neuropathic feeling in her hands and cold sensitivity, adding that the symptoms "had escalated over the last 2 weeks," requiring her to medicate. *Id*.

On exam, Petitioner displayed thickened lower extremities with non-pitting edema, as well as mild discomfort in the lumbar spine and a chemotherapy-induced rash on her arms. Ex. 3 at 29. Dr. Schwartz ultimately characterized her symptoms as "very consistent with chemotherapy effect." *Id.* He predicted that things should "markedly improve once chemotherapy gets out of her system over the next few weeks," noting that she was scheduled to begin six weeks of radiation therapy in January 2013, followed by adjuvant therapy with an oral chemotherapy medication. *Id.*

A week later, however, Ms. Rushing returned to Dr. Schwartz's office on December 13, 2012, for an unscheduled sick visit. Ex. 3 at 25. She now complained of more persistent bone pain, particularly in her legs and left hip (and extending to her left buttock), that she reported made sleep difficult. *Id*. Her bloodwork was normal, as was a hip x-ray. *Id*. at 19–24. She also reported constipation and a rash on her arms, plus concerns about loss of appetite. *Id.* Exam revealed mild

hypotension, and she was given one liter of IV saline solution, anti-nausea medication, and a corticosteroid. *Id*. at 26.

On December 17, 2012, Petitioner's husband called Dr. Schwartz's office, communicating his concerns about numbness and tingling she was experiencing in her hands, feet, and legs. Ex. 3 at 18. Petitioner was again advised that the chemotherapy side effects would "slowly resolve," but she decided to try a medication for relief of neuropathic pain. *Id*. Her husband called back two days later, however, now reporting that Petitioner's generalized weakness and nutritional status had resulted in her falling down at home. *Id*. at 17. (The nurse who documented the call also memorialized the fact that Petitioner had reported feeling well the previous day). *Id.*

On December 21, 2012, Petitioner returned to Dr. Schwartz for treatment associated with "complications of Taxol therapy." Ex. 3 at 13. Dr. Schwartz recorded his impression that she now looked quite weak, and she informed him she had experienced several falls over the prior week due to pain and instability. *Id.* She also reported increasing numbness in her feet and ankles, making it difficult to button or zip her clothing, that she felt "freezing all the time," especially in her fingers and toes, and that she felt a brief electric-shock sensation whenever her arms were touched. *Id*. She also could not walk independently and was using a walker. *Id*. On exam, she had intact sensation in her lower extremities and preserved plantar flexion, but decreased dorsiflexion in both lower extremities, as well as reduced hip flexor strength, particularly on the left side. *Id.* at 14. Her most recent lab work was normal. *Id.*

Based on the foregoing, Dr. Schwartz opined that Petitioner's neuropathy had "taken a rapid turn for the worse," and he deemed it a grade three neuropathy of the upper extremities. Ex. 3 at 14. He continued to propose she receive medication for neuropathic pain, Gabapentin, even though he noted it might contribute to her hypotension and instability. *Id*. He also prescribed a five-day course of prednisone to address her left buttock pain (adding that a longer course might be warranted if the cause of her symptoms was polymyalgia rheumatica), and decided she should not at that time start radiation therapy—but otherwise did not note any view that her symptoms reflected possible GBS. *Id.*

Several days later (December 26th), Petitioner was again in Dr. Schwartz's office, and was seen at this time by Nurse Evans. Ex. 3 at 7. She complained of left eye redness and swelling, along with leg swelling that caused a tight feeling, particularly in her ankles. *Id*. She also described a fall two days earlier while she was trying to navigate two stairs in her home, that she felt as though her legs might give out, and continued to experience numbness and tingling along the solar aspect of both feet (although it was at least "no worse than previous" if not better). *Id*. Exam revealed the existence of possible conjunctivitis, and the leg swelling was proposed by Nurse Evans to be a side effect of the steroidal medications Petitioner was receiving. *Id.* at 7, 8. But Nurse Evans ordered additional bloodwork and helped schedule Petitioner for a visit with a neurologist

in mid-January 2013. *Id.* (Before leaving this treatment visit, Petitioner fell while in the bathroom and fractured her ankle, resulting in an emergency room visit. Ex. 4 at 9465, 9975–78. She later that same month required an orthopedic procedure for ankle repair. *Id.* at 9446, 9469, 9881–83).

Treatment for Neurologic Issues in 2013

On January 2, 2013, Petitioner was readmitted to the hospital on the advice of Dr. Schwartz's office due to her ongoing and unresolved symptoms. Ex. 4 at 9465. It was observed that she had become weaker after her ankle surgery, and that her husband's own physical maladies made it difficult for him to care for her. *Id*. at 9465–66. She was also deemed by Nurse Evans to have developed some kind of neuropathy with weakness "during chemotherapy; significantly more so after her last dose." *Id*. at 9467. Petitioner informed treaters that although her pain levels had improved, she felt tired and weak, and was still experiencing numbness and tingling in her hands and feet. *Id.* at 9466. On exam, Petitioner again displayed pitting edema of the legs. Ex. 4 at 9467. She also displayed good upper extremity grip strength, strong right leg strength, along with weak left leg strength. *Id.* at 9511. But a whole-body bone scan ordered for her during her hospitalization was negative for malignancy. *Id.* at 9784.

Petitioner was subsequently discharged to a skilled nursing facility with the diagnosis of failure to thrive, and she remained at the facility through the middle of January 2013. Ex. 4 at 9789; *see generally* Ex. 5. During this timeframe, on January 15, 2013 (now eight weeks post-vaccination), Petitioner underwent a lumbar MRI and lumbar puncture at the direction of neurologist David Schmidt, M.D. Ex. 6 at 111, 114. The MRI revealed the existence of lumbar spondylosis causing mild to moderate stenosis at levels L2-3, L3-4, and L4-5. *Id.* at 111. The lumbar puncture revealed an elevated cerebrospinal fluid ("CSF") protein level of 94 (range 15-45). *Id.* at 114. No specific diagnosis was recorded or treatment ordered in reaction to these findings.

Within a week of discharge from the skilled nursing facility, Petitioner was readmitted to the hospital after complaining of lower extremity weakness. Ex. 5 at 56; Ex. 6 at 7. She was examined on admission by family physician Jonathan Shraga, M.D. Ex. 6 at 7. Dr. Shraga notes in the relevant record from this visit that he had discussed Petitioner's course with Dr. Schwartz, who explained to Dr. Shraga that he had initially attributed her neuropathy to her chemotherapy, but now felt that "[chemotherapy] not [] contributing *at this time*." *Id*. (emphasis added). The history section for this record also noted that Petitioner had received an influenza vaccination in November. *Id*. Petitioner informed treaters at this time that she continued to experience ongoing bilateral finger tingling, although her lower extremities were her primary concern. Ex. 6 at 7. On exam, she displayed preserved upper extremity strength and intact sensation, but decreased strength and intact sensation in her lower extremities, trace reflexes in the left knee, and absent

reflexes in the right knee. *Id*. at 9. Dr. Shraga's concern was the possibility that Petitioner had an inflammatory demyelinating polyneuropathy. *Id*.

Dr. Shraga ordered a neurology consult, and later that month (January 22, 2013), Petitioner underwent an EMG[4]/NCS[5] conducted by neuromuscular specialist, Dr. Francois Picot. Ex. 44 at 2–3. Dr. Picot interpreted the study results as consistent with an axonal polyradiculopathy. *Id.* at 3. He could not rule out a lumbosacral plexopathy or a diffuse lower extremity polyradiculopathy, however. *Id.* at 3. The same day, Petitioner again saw Dr. Schwartz, who now deemed her condition to be consistent with GBS—adding that she had received the flu vaccine in November. Ex. 6 at 67. Ms. Rushing that same month received a five-day course of IVIG.[6] *Id.* at 69. But it did not improve her leg symptoms or function. *Id.* at 38–39. On January 23, 2013, Petitioner underwent a repeat lumbar puncture, which revealed an elevated CSF protein level of 79. Ex. 4 at 9301.

On January 28, 2013, Dr. Schwartz memorialized in a record the fact that he had discussed Petitioner's potential polyradiculopathy diagnosis with another treater that morning. Ex. 4 at 9281. Dr. Schwartz noted a lack of response to IVIG, and that the other treater's "informal opinion is the current findings are not classic for GBS and more likely a GB-like syndrome that may be post-viral." *Id.* However, Dr. Schwartz also proposed that the likelihood Petitioner's neuropathic symptoms, and worsening course, were attributable to her prior Taxol treatment to be "extremely rare esp[ecially] without upper extremity changes." *Id*. Dr. Schwartz ultimately opined that "no clear etiology w[ould] be found," and questioned whether an underlying inflammatory process was causing her radiculopathy. *Id*. at 9282.

During a visit with Dr. Picot on January 28th, Petitioner displayed absent reflexes, some upper extremity weakness, limited leg strength, and reduced sensation in mid-thigh and left knee. Ex. 6 at 10, 12. But, like Dr. Schwartz, Dr. Picot had trouble pinpointing the best diagnostic explanation for Petitioner's symptoms. Thus, although "[s]he has been diagnosed with the possibility of having the axonal form of Guillain-Barre syndrome," he could not "rule out the possibility, in addition to a possible polyneuroradiculopathy, a possible lumbosacral

[4] Electromyography is the process by which "an electrodiagnostic technique for recording the extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions, and during electrical stimulation; performed using any of a variety of surface electrodes, needle electrodes, and devices for amplifying, transmitting, and recording the signals." *Electromyography*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=15854&searchterm=electromyography (last visited Feb. 16, 2024).

[5] A nerve conduction study measures the amount and speed of conduction of an electrical impulse through a nerve to determine nerve damage and destruction. *Nerve Conduction Studies*, Health Library, Johns Hopkins Medicine, https://www.hopkinsmedicine.org/health/treatment-tests-and-therapies/nerve-conduction-studies (last visited Feb. 16, 2024).

[6] Intravenous immunoglobulin ("IVIG") is a blood product used to treat patients with antibody deficiencies, including neurological disorders. Clinical Use of Intravenous Immunoglobulin, NCBI (2005) https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1809480/ (last visited on Feb.1 6, 2024).

polyradiculopathy," or "the effects of chemotherapy," especially given the overall mix of presenting symptoms and test findings. *Id.* at 12. Dr. Picot also noted that a "paraneoplastic process" attributable to her prior breast cancer might be explanatory. *Id.* And although "[f]lu shots have been associated" with GBS, her symptoms would reflect an "atypical presentation" for a vaccine-caused neuropathy given the attenuated timeframe between onset and vaccination. *Id.*

Dr. Schwartz had another office visit with Petitioner on January 30, 2013, just prior to her hospitalization discharge. Ex. 4 at 9271. At this time, she displayed no upper extremity symptoms, plus very slight improvement in the movement of her right leg, and Dr. Schwartz proposed decreasing her neuropathic pain medication dose. *Id.* Upon discharge to an inpatient rehabilitation facility, Petitioner's differential diagnosis included axonal GBS versus polyneural radiculopathy or lumbosacral polyradiculopathy. Ex. 6 at 5.

Petitioner remained in this rehab facility for approximately five weeks, or until March 8, 2013. Ex. 4 at 9438. In the course of the stay, urinary retention resulted in Petitioner being diagnosed with neurogenic bladder requiring intermittent catheterization, but otherwise she experienced increased mobility and functionality. *Id.* at 9438–39. She saw Dr. Schwartz again after discharge, and he now expressed less confidence in the possibility that her symptoms were the by-product of chemotherapy. Ex. 13 at 46. Dr. Schwartz noted that Petitioner's "paraneoplastic panel" was negative and that "[t]he best explanation at this time [for her polyradiculopathy] is possibly connected with her influenza vaccine that was given in late November versus an idiopathic event." *Id.* at 47.

Thereafter, From May 22 through December 18, 2013, Ms. Rushing attended thirty-five outpatient physical therapy ("PT") sessions. Ex. 4 at 146–279. The course of therapy was helpful, and by its conclusion she no longer needed a wheelchair and also displayed improved lower extremity strength. On October 13, 2013, she followed up with Dr. Schwartz, and although she still had neuropathy affecting her hands and feet, as well as lower extremity weakness, she was ambulating well with a walker. Ex. 7 at 61.

Subsequent Treatment

Petitioner has filed records of her treatment and health history after January 2014, but they mostly illuminate her subsequent progress rather than provide diagnostic explanations for her post-vaccination condition. Thus, she showed some recovery in follow-up visits with Dr. Picot in February 2014 (despite ongoing foot weakness and pain). Ex. 8 at 3, 5. She ceased neurologic treatment by July 2015. Ex. 7 at 124. And although she was hospitalized in November 2017 (now *five* years post-vaccination) after experiencing weakness, and a treater opined she might be displaying deconditioning versus chronic inflammatory demyelinating polyneuropathy ("CIDP"),

no confirmation of the CIDP diagnosis was obtained and she was discharged with the diagnosis listed as "weakness likely 2/2 deconditioning, shingles." Ex. 10 at 23, 24, 26.

II. Expert Reports & Treater Statements

A. *Treater Statements*

1. Dr. Francois J. Picot – Dr. Picot was one of Ms. Rushing's neurologic treaters, and he has offered in this case several witness statements in support of the claim. *See* Statement, dated July 31, 2020, filed as Ex. 16 (ECF No. 26-1) ("First Picot Statement"); Statement, dated Oct. 26, 2022, filed as Ex. 70 (ECF No. 50-1) ("Second Picot Statement"). None of the statements are notarized, nor do they meet the requirements of a sworn declaration under federal law. 28 U.S.C.A. § 1746.

Dr. Picot's second statement sets forth his general medical credentials. He is a neuromuscular specialist with Atrium Health Neurology in Huntersville, North Carolina. Second Picot Statement at 1. He received his medical degree from Tulane University. *Id.* Thereafter, Dr. Picot completed his residency at the University of Maryland, followed by a fellowship at the State University of New York. *Id.* Dr. Picot's practice involves treating patients with neurological disorders, including GBS. *Id.*

As both statements represent (although the second in somewhat greater detail than the first), Dr. Picot treated Petitioner in January 2013, seeing her again in February 2014. First Picot Statement at 1; Second Picot Statement at 1. He deems her GBS to have been an "axonal form." Second Picot Statement at 1. In support, he references the EMG he performed, adding that its results "would not be expected for neuropathy from Taxol treatment." *Id.* He also represents she first experienced neurologic symptoms associated with GBS in early December 2012. *Id.*

Dr. Picot opines that Petitioner's November 2012 vaccination played some substantial role in Petitioner's GBS. However, his first statement sets forth this opinion in wholly conclusory form, with no explanation for his reasoning. *See* First Picot Statement at 1. The second statement makes clear, however, that his causation views are based on review of, and concurrence with, the treater reports of Dr. Schwartz plus Dr. Steinman's immunology opinion. Second Picot Statement at 1. Dr. Picot's second statement also opines that he deemed alternative causation explanations, like effects of Taxol, unpersuasive, since Petitioner received only a low dose of it (with no greater evidence that this dosage level had ever caused injury in a comparable patient), and because she "had no use of either leg" as of her late-January 2013 hospitalization. *Id.* Otherwise, he opined that her EMG results were inconsistent with a Taxol-related neuropathy. *Id.*

2. Dr. Garry Schwartz – Dr. Schwartz, Petitioner's oncologist treater, offered several statements and reports to support the claim. *See* Statement, dated July 27, 2020, filed as Ex. 15 (ECF No. 24-1) ("First Schwartz Statement"); Statement, dated August 22, 2022, filed as Ex. 43 (ECF No. 42-1); Statement, dated September 7, 2022, filed as Ex. 60 (ECF No. 48-1)

("Schwartz Rep."). Like Dr. Picot's contributions, none are sworn statements, but the third submission contains annotations that make it more of an expert report (even if offered by one of Petitioner's actual treaters). Petitioner has not filed a CV for Dr. Schwartz.

First Statement

Dr. Schwartz's first statement contains his review of Petitioner's medical history, based on his direct experience treating her. He notes that she had initially been diagnosed with breast cancer in the spring of 2012, and later received chemotherapy—including "12 doses of weekly Taxol" in the fall of 2012, ending December 7, 2012. First Schwartz Rep. at 1. He notes that he saw Petitioner in mid-October of that year, and although she made some complaints about fatigue and extremity discomfort (and even reported armpit numbness that radiated to her hand), her issues predated chemotherapy, and were not otherwise consistent with neuropathy. *Id.*

Petitioner next reported hand neuropathy from her Taxol treatments on November 30, 2012 (and thus after vaccination), and Dr. Schwartz (admitting that "[p]eripheral neuropathy is a well-recognized and common side effect of Taxol") characterized these complaints as Taxol-associated. First Schwartz Statement at 1, 2. However, the lower extremity complaints she later lodged on December 7th were in his assessment new, and distinguishable from the more minor neuropathic issues she had reported a week before. *Id.* at 2. In particular, the cold sensitivity she was experiencing was far more characteristic of Taxol-associated side effects than the new issues.

Later that December, Petitioner began to report leg and feet numbness and tingling—another new symptom different from the more minor hand neuropathy. First Schwartz Rep. at 2. And then she and her husband started to report falls, which Dr. Schwartz did not at all view as consistent with "resolving chemotherapy side effects." *Id.* This, plus the worsening of other lower extremity symptoms, led Dr. Schwartz to conclude that Petitioner's presentation could not be chemotherapy-associated. *Id.* at 2–3. And by January, when Petitioner was hospitalized, Dr. Schwartz was comfortable sharing his view about the nature and cause of her symptoms with treaters like Dr. Shraga. *Id.* at 3. He was thus ultimately comfortable with the conclusion (which he noted when he next saw Petitioner in March 2013) that Petitioner's symptoms overall were not likely due to chemotherapy (even if the initial symptoms were). *Id.* at 4.

Second Statement

Dr. Schwartz's second statement is considerably more succinct than the first, and merely provides his reaction to the expert reports filed as of that date. *See* Second Schwartz Statement at 1. He agrees with Dr. Steinman's causation report over what Respondent had offered. He also reiterates the view that Petitioner's presentation in December 2012 was "far more consistent with vaccine-induced GBS," even if "Taxol-induced neuropathy" is more commonly observed. *Id.* In support, Dr. Schwartz noted that of the "many hundreds" of his patients who have received Taxol, he had never had a patient with the "degree of neuropathy" displayed by Ms. Rushing. *Id.*

Third Statement

Dr. Schwartz's final statement reads more like a full expert report and is supplemented by nine literature cites. Schwartz Rep. at 6. In it, he attempts to provide a more detailed explanation for why the dosage of Taxol received by Petitioner could not have caused the degree of neuropathy she experienced, despite the opinions of Respondent's experts.

Dr. Schwartz emphasized that toxicity to chemotherapy was "dose dependent," and noted that Petitioner had received what he considered a "relatively low dose" of Taxol—80 mg/m^2 weekly, for a one-hour period, for a total of twelve weeks—meaning she had received cumulatively 960 mg/m^2. Third Schwartz Statement at 1. But this was a significantly lower dosage than what the patient subjects had received in studies referenced by Drs. Callaghan and McClain. One such study sought to compare the antitumor effectiveness of Taxol against a different chemotherapy drug. S. Jones et al., *Randomized Phase III Study of Docetaxel Compared with Paclitaxel in Metastatic Breast Cancer,* 23 J. Clin. Oncol. 5542 (2020), filed as Ex. C Tab 2 (ECF No. 34-3) ("Jones").

Jones (like other studies discussed herein) evaluated the known neuropathic impact of two comparable chemotherapeutic agents: Taxol (the marketing name for paclitaxel), and docetaxel (a differently-formulated, stronger agent). Jones at 5542–43. It attempted to do so, however, via a more scientifically-precise, randomized trial, evaluating over 400 breast cancer patients in North America who received the two evaluated therapies. *Id.* at 5544. 224 subjects received Taxol—and the study participants therein received, as a median, four cycles of treatment up to a maximum of 35, with a mean dose per cycle of 173 mg/m^2. *Id.* This meant, Dr. Schwartz maintained, that some studied individuals received cumulative doses up to 6,125 mg/m^2, with no patients receiving doses in the more limited range Petitioner did. Third Schwartz Statement at 1. Otherwise, Jones observed far more instances of neurosensory than neuromotor adverse events from Taxol (59 percent of the 209 subjects who experienced any adverse event versus 12.6 percent), and the percentage of severe neuromotor events was even lower. Jones at 5548.

The same dosage disparity in causing severe motor neuropathies was observed in another, earlier article evaluating comparative effectiveness of different chemotherapy treatments. R. Freilich et al., *Motor Neuropathy Due to Docetaxel and Paclitaxel,* 47 Neurol. 115 (1996), filed as Ex. A Tab 1 (ECF No. 29-2) ("Freilich"). Freilich endeavored (like Jones, but well before it) to compare the neuropathic effects of Taxol against docetaxel. Only four of the 64 evaluated patients received Taxol, and each of the four received no less than 250 mg/m^2 at a time, for a cumulative dose of up to 6,250 mg/m^2. Freilich at 116. Although all four experienced weakness eventually progressing to motor symptoms as the cumulative dosage increased, even the subject receiving the highest dose maintained the ability to ambulate, and no subjects experienced decreases in strength in the one to two-month period after cessation of the chemotherapy. Freilich at 117–18. This was contrary to Ms. Rushing's experience. Third Schwartz Statement at 1–2.

Dr. Schwartz noted other distinctions between Petitioner's experience and the subjects in Respondent's cited studies. Third Schwartz Statement at 2. For example, although Respondent's experts maintained that Freilich underscored the association between Taxol-associated severe neuropathies and elevated protein levels observed in CNS testing, this was based on data for only two subjects—only one of whom had in fact received Taxol (and at a significantly higher dose as well). Freilich at 117. Otherwise, none of Respondent's studies involved an individual who experienced a stage 4 motor neuropathy after receipt of a Taxol dosage comparable to Petitioner's—and where symptoms worsened even after treatment concluded. Third Schwartz Statement at 2.

A study discussed only by Dr. McClain was also, in Dr. Schwartz's estimation, unhelpful to Respondent. Third Schwartz Statement at 3; C. Scripture et al., *Peripheral Neuropathy Induced by Paclitaxel: Recent Insights and Future Perspectives,* 4 Current Neuropharm. 165 (2006), filed as Ex. C Tab 1 (ECF No. 34-2) ("Scripture"). Scripture, a review article, discusses the then-current state of scientific and medical views about Taxol's known neuropathic side-effects and thinking about how to mitigate them. In particular, Scripture (a) confirms that a sensory neuropathy is the most common side-effect, and (b) that this adverse effect is "dose- and infusion-duration related, and most frequently occurs when the dose . . . exceeds 250 mg/m^2 infused over [more than] 24 hours." Scripture at 166, 167 (lower dosages per cycle associated with milder neuropathic symptoms, which might not manifest until cumulative dosage exceeded 1,400 mg/m^2). Thus, in Dr. Schwartz's reading, Scripture confirmed not only that Taxol was associated only with a more limited, sensory-style neuropathy, but that dosage mattered—and that the evidence referenced in Scripture for the latter point came from the Freilich article. Third Schwartz Statement at 3. Only very high dosages not relevant to Petitioner's experience—more than 250 mg/m^2 in a cycle—could a more severe, motor neuropathy occur. *Id.*

Other studies not mentioned by Respondent's experts were, Dr. Schwartz maintained, fully consistent with his opinion about the dosage-severity relationship. One study considered the effect of Taxol chemotherapy treatments received by 105 breast cancer patients over a 12-week period. H. Timmins et al., *Weekly Paclitaxel-Induced Neurotoxicity in Breast Cancer: Outcomes and Dose Response*, 26 The Oncologist 336 (2021), filed as Ex. 65 (ECF No. 49-5) ("Timmins"). The dosages utilized therein were comparable to what Petitioner had received (80 mg/m^2 at a single weekly administration), and the patients experienced only the kind of more mild, sensory neuropathic symptoms Petitioner first experienced (even if Timmons does also emphasize the degree to which chemotherapy-induced neuropathic symptoms generally do not vanish once treatment ends, but in fact can linger months beyond). Timmins at 367, 368–69. Although symptoms severity did increase as the cumulative dosage went up, "no patient was non-ambulatory, paralyzed, or required hospitalization." Third Schwartz Statement at 4.

Overall, many studies confirmed the association between dosage and degree of neuropathic symptoms. *See, e.g.*, M. Green et al., *Weekly Paclitaxel Improves Pathologic Complete Remission in Operable Breast Cancer When Compared With Paclitaxel Once Every Three Weeks,* 23 J. Clin.

Oncol. 5983 (2022), filed as Ex. 66 (ECF No. 49-6), at 5990 ("[Taxol] administered at a lower dose of 80 mg/m was very well tolerated, with a reduced risk of neutropenic fever and grade 3 neuropathy, compared with once-every-three weeks [Taxol]"). One review article, compiling the results of 14 clinical trials, confirmed that severe, motor-oriented neuropathies were exceedingly uncommon, even at doses *higher* than what Petitioner had received. Third Schwartz Statement at 4–5; E. Rivera and M. Cianfrocca, *Overview of Neuropathy Associated with Taxanes for the Treatment of Metastatic Breast Cancer*, 75 Cancer Chemother. Pharmacol. 659 (2015), filed as Ex. F Tab 1 (51-6) at 661 Table 1 (reviewing results of 11 studies), 662.

B. *Petitioner's Experts*

1. Dr. Lawrence Steinman – Dr. Steinman, a neurologist, prepared two written reports for the Petitioner. Report, dated August 16, 2022, filed as Ex. 42 (ECF No. 41-1) ("First Steinman Rep."); Report, dated February 13, 2023, filed as Ex. 71 (ECF No. 55-1) ("Steinman Second Rep."). Dr. Steinman opines that the flu vaccine likely caused Petitioner's neuropathy, which is best understood to be GBS, and that her symptoms began in a timeframe consistent with the Table claim's requirements.

As shown in his CV, Dr. Steinman received his undergraduate degree from Dartmouth College, and his medical degree from Harvard Medical School. *Curriculum Vitae,* filed as Ex. 45 on Aug. 23, 2022 (ECF No. 44-1) ("Steinman CV") at 1. He then completed residencies in neurology and pediatrics at Stanford University. *Id.* He has worked as a professor of neurology and pediatrics at Stanford for the past 41 years. *Id.*; Steinman First Rep. at 2. He is board certified in neurology from the American Board of Psychiatry and Neurology. Steinman CV at 2. Dr. Steinman has also published hundreds of peer-reviewed publications on neurology and autoimmune disease. *Id.* at 5–49. He holds several patents related to the diagnosis and treatment of autoimmune and demyelinating diseases. *Id.* at 2–3. He presently serves as the George A. Zimmerman Professor of Neurological Sciences, Neurology, Genetics and Pediatrics at Stanford University. *Id.* at 1.

First Report

Much of Dr. Steinman's first report includes the kind of "boilerplate" statements about his expertise that he offers in almost all Vaccine Program cases in which he is retained as an expert. *See, e.g.*, First Steinman Rep. at 1–3. He also provided a medical records summary, although it is consistent with what is set forth above in this decision. *Id.* at 3–9. Thus, only a few pages of this report are germane to the present dispute. *Id.* at 9–11.

The heart of Dr. Steinman's opinion begins with his explanation of Taxol's components and how they function. Taxol, he stated, is an "antineoplastic agent" that can (as a side effect to its intended chemotherapy purpose) also "promote[] the formation of abnormal bundles of

microtubules within the cytoplasm," leading to neuropathy from the dysfunction of those microtubules. First Steinman Rep. at 9; *see also* Scripture at 165. Thus, the drug's intended impact on microtubules could also cause nerve harm—a well-understood risk factor for the chemotherapeutic agent.

In most cases, Taxol produces sensory peripheral neuropathies that are "dose- and infusion-duration related." First Steinman Rep. at 9. By contrast, motor neuropathies are far less common in patients receiving Taxol, except in instances involving higher doses. *Id.* Dr. Steinman emphasized that although some articles (Freilich, for example) observed instances of motor neuropathies in Taxol-receiving patients, they commonly remained ambulatory—which was not in this case true of the Petitioner. *Id.* In addition, such literature noted the association between cessation of the Taxol treatments and symptoms improvement—again, something not observed in Petitioner's case (since more than a month separated the end of her treatment and the acute symptoms leading her to seek medical intervention in late-January 2013). First Steinman Rep. at 9, 11; *see also* V. Chaudhry et al., *Peripheral Neuropathy From Taxol and Cisplantin Combination Chemotherapy: Clinical and Electrophysiological Studies,* 35 Ann. Neurol. 304 (1994), filed as Ex. 57 (ECF No. 44-13), at 306 (out of 21 studied patients receiving Taxol, three-quarters experienced sensory neuropathies, beginning sooner after administration of larger doses; axonal neuropathies occurred in some patients, but severity was correlated with cumulative dose amounts).

Dr. Steinman concurred with the diagnosis in this case of axonal GBS, noting that the medical records provided support for it—evidence of the elevated CSF protein levels, along with observed instances by Dr. Picot of lower extremity weakness. First Steinman Rep. at 10. Her overall experience with the monophasic progression of the disease was also consistent with the diagnosis—plus her nadir within 28 days of the proposed December 7th onset, since in late December she experienced the fall leading to the breaking of her ankle, and worsening of weakness that encouraged treaters to pursue a lumbar puncture and MRI. *Id.* The lack of symptoms responsiveness to IVIG was not significant to Dr. Steinman, since (as Dr. Picot had proposed) it had likely been administered too late in her treatment course (when measured from onset).

Dr. Steinman accepted onset as beginning around the time of Petitioner's complaints of lower extremity pain on December 7, 2012—within the 3–42 day timeframe for onset, based on the November 20th vaccination. First Steinman Rep. at 10. He admitted that around this time, she had also been experiencing the more "recognized" kind of Taxol-associated sensory neuropathic symptoms (her fingertip sensations in particular), but deemed them distinguishable from the GBS symptoms more likely caused by vaccination. *Id.* In so opining, Dr. Steinman disagreed with Dr. Callaghan's view that onset occurred in early November 2012 (meaning, if correct, that a nadir almost two months later was inconsistent with GBS's usual course). *Id.* at 11. The record, he

maintained, showed only sensory-type symptoms in November, with no actual GBS-oriented symptoms before early December. *Id.*

Dr. Steinman also reacted to other points raised in the initial reports of Respondent's experts. For example, he deemed them to give insufficient weight to the treater opinions of Drs. Schwartz and Picot, who had seen Petitioner at the time of her initial presenting symptoms. First Steinman Rep. at 11.

Second Report

Dr. Steinman's second report reacted solely to assertions made in Dr. Callaghan's second report—and his acknowledged focus was the "narrow issue" of the GBS diagnosis and onset—although he began by addressing the argument that Petitioner's Taxol treatment explained her injury. Second Steinman Rep. at 1. Thus, although Dr. Callaghan had contended that many instances of Taxol-induced neuropathy had *some* motor neuropathy features, such as leg weakness, Petitioner's presentation was characterized by a more severe form of motor neuropathy, which was far less common in cases attributable to the chemotherapy. *Id.* And the medical records revealed Petitioner's own "instability and pain" had been severe enough in late-December 2012 to cause her to fall several times—while at the same time her other neuropathic symptoms broadened and increased. *Id.* A subsequent fall required surgery, and then continued symptoms progression led to diagnostic testing (an MRI and lumbar puncture) that seemed to confirm GBS. *Id.* at 2.

Thereafter, other treaters who saw Petitioner in late-January 2013 noted that the severity of Petitioner's symptoms was not consistent with a Taxol-caused form of neuropathy. Second Steinman Rep. at 3–4. Eventually, after hospitalization and associated testing Petitioner was diagnosed with an axonal form of GBS. While the roles of Taxol versus the flu vaccine did not, in Dr. Steinman's estimation, present an "either/or proposition," since both could cause neuropathic symptoms, the evidence in his view did support the vaccine as causal, given treater statements—even if Taxol could result in neurologic symptoms as well. *Id.* at 4–5.

2. Dr. M. Eric Gershwin – Dr. Gershwin is an immunologist and rheumatologist, and he prepared a single report for Petitioner. Report, dated March 25, 2021, filed as Ex. 17 (ECF No. 31-1) ("Gershwin Rep."). Dr. Gershwin proposed that the flu vaccine caused Petitioner's GBS, and in a medically acceptable timeframe as well.

Dr. Gershwin received his undergraduate degree from Syracuse University, his medical degree from Stanford University, and his graduate degree from the Centre for Astrophysics and Supercomputing. *See* Curriculum Vitae, filed Mar. 30, 2021 (ECF No. 21-25) ("Gershwin CV") at 1. He completed his internship and residency at Tufts New England Medical Center, and two fellowships in rheumatology and allergy/immunology at the National Institutes of Health. *Id.* at 2. He is currently employed as Director of the Allergy-Clinical Immunology Program and Professor

of Medicine at University of California at Davis ("UC Davis"). *Id.* at 1–2. Dr. Gershwin is licensed to practice medicine in California and is board certified in allergy and immunology, rheumatology, and internal medicine. *Id.* at 2. He also publishes extensively and is well researched in epidemiological studies. *Id.*

Dr. Gershwin included his own review of Petitioner's history before commencing with his opinion. Gershwin Rep. at 1–2. (He also provided a summary of his qualifications. *Id.* at 3). He deemed her history "long and complicated," but demurred from offering his own take on her proper diagnosis, focusing instead on the role the flu vaccine could have played from a causation standpoint. *Id.* at 2. He also offered a lengthy explanation of GBS generally and its known etiologies (although I do not deem this matter to be contested, other than to the extent it bears on the Taxol-causation question). *Id.* at 4–11.[7] One immediate point Dr. Gershwin made, however, was to distinguish Petitioner's "Taxol-related neuropathy"—which he proposed had likely begun pre-vaccination—from her "GBS-related neuropathic symptoms." Gershwin Rep. at 2. Then, after explaining how vaccines generally prompt an immune reaction, Dr. Gershwin opined that "GBS is an environmentally induced autoimmune disease," and that the onset of Petitioner's GBS-like symptoms arose in a medically acceptable timeframe measured from vaccination. *Id.* at 15.

C. *Respondent's Experts*

1. Dr. Brian Callaghan – Dr. Callaghan, a neurologist and academic, prepared two expert reports on Respondent's behalf. Report, dated October 5, 2020, filed as Ex. A (ECF No. 29-1) ("First Callaghan Rep."); Report, dated December 30, 2022, filed as Ex. E (ECF No. 51-1) ("Second Callaghan Rep."). Dr Callaghan opines that Petitioner experienced a sensory and motor neuropathy attributable only to her Taxol treatment.

Dr. Callaghan received his undergraduate degree from the University of Michigan, his medical degree from the University of Pennsylvania in 2004, and his master's in science from the University of Michigan in 2011. *Curriculum Vitae*, filed as Exhibit B (ECF No. 29-6) ("Callaghan CV") at 1. He is board certified in psychiatry and neurology, as well as electrodiagnostic medicine. *Id.* Dr. Callaghan was appointed to be a clinical lecturer at the University of Michigan Health System's Department of Neurology in 2009 and has been an Associate Professor of Neurology there since 2018. *Id.* He has published more than 100 articles and medical book chapters, most of which focus on neuropathies, and his research interest lies in diagnostic evaluation and testing of peripheral neuropathies. First Callaghan Rep. at 1; Callaghan CV at 2, 11–20. Dr. Callaghan reports that he encounters and treats approximately five patients with GBS per year, and five patients with chemotherapy-induced neuropathy per year. First Callaghan Rep. at 1.

[7] These points are in fact almost superfluous in this case, especially since it involves a Table injury, where causation is presumed.

First Report

Dr. Callaghan's initial report was based on his review of Petitioner's medical history, plus his own desktop research (performed solely for this case) into "the association of vaccines and paclitaxel (Taxol) with axonal neuropathy." First Callaghan Rep. at 1, 2–3. He had also had the benefit of having reviewed the contents of Drs. Picot and Schwartz's first letter-statements. *Id.* at 3.

Regarding the medical record, Dr. Callaghan emphasized that Petitioner's first symptoms were limited to fingertip numbness (even though her symptoms progressed to more widespread numbness and tingling plus lower extremity weakness). First Callaghan Rep. at 3. He also noted that Dr. Schwartz himself had initially deemed her presentation to be consistent with a Taxol-caused sensory neuropathy, and that other testing conducted during the January 2013 hospitalization (MRIs, a lumbar puncture) produced unrevealing results—plus IVIG had not proven beneficial. *Id.* at 3–4. Because Dr. Callaghan had not received record evidence of EMG testing results, he did not opine on what they showed specifically (although he acknowledged that records indicated they were consistent with a polyradiculopathy). *Id.*

Petitioner's pre-vaccination Taxol treatments were, in Dr. Callaghan's estimation, "[b]y far the most likely cause" for her sensory neuropathy. First Callaghan Rep. at 4. He noted that certain literature established Taxol to be a "well-known cause of sensory neuropathy" in patients receiving this form of chemotherapy. *Id.* Motor neuropathy could also be a side effect of Taxol, and Dr. Callaghan noted that (in his view) the chance of a "severe grade" form of motor neuropathy was higher in individuals (like Petitioner) who were already suffering from a higher grade of sensory neuropathy. *Id.* He based this contention, however, on the findings from Jones, which were not only specific solely to the set of 200-plus studied individuals, but found severe forms of *any* neuropathy, motor or not, to be far less common than a milder form. *See* Jones at 5548, Table 5.

GBS caused by the flu vaccine, by contrast, was less likely. As a general matter, flu vaccine-caused GBS was an unlikely occurrence. First Callaghan Rep. at 4. Since axonal neuropathies constituted less than a fifth of all GBS cases in North America, that form was even less common. S. Kuwabara & N. Yuki, *Axonal Guillain-Barré Syndrome: Concepts and Controversies,* 12 Lancet Neurol. 1180 (2013), filed as Ex. A Tab 4 (ECF No. 29-5) ("Kuwabara") at 1181. Given the higher association between Taxol and neuropathies (in comparison to the rarity of vaccine-caused GBS), it seemed most likely to Dr. Callaghan that Petitioner's Taxol treatments were the cause of her injury.

Dr. Callaghan in fact did not find persuasive the conclusion that Petitioner had experienced GBS in the first place. He noted that although patients with axonal GBS usually display preserved reflexes, Ms. Rushing had absent reflexes, a feature of more common forms of GBS. First

Callaghan Rep. at 3; Kuwabara at 1182. In addition, the timeframe from Petitioner's neurologic symptoms onset in early November 2012 and her nadir (which he deemed to have occurred during her January 2013 hospitalization) was more than two months—inconsistent with GBS (which should see nadir between 12 hours and 28 days of onset).[8]

Finally, Dr. Callaghan disputed Dr. Schwartz's suggestion that Petitioner had experienced *both* a Taxol-induced sensory neuropathy plus axonal GBS. First Callaghan Rep. at 3. In support, he argued that the EMG evidence of axonal "changes" was as consistent with GBS and with a Taxol-associated neuropathy (although as noted he had not reviewed the EMG results directly). *Id.* The elevated protein levels in CSF testing were also not indicative of GBS, since they "only indicate[] that the injury occurred at the nerve root level," and that otherwise such elevated levels were a "known finding" for Taxol neuropathies. *See, e.g.*, Freilich at 117 (elevated protein was the sole abnormality seen in the two patients whose CSF was tested—although Freilich does not otherwise comment on the association between this GBS indicator and Taxol). And he again emphasized how (in his view) robust the evidence was supporting a Taxol-neuropathy association. First Callaghan Rep. at 3.

Second Report

The second expert report submitted by Dr. Callaghan responded to the additional witness statements provided by Drs. Picot and Schwartz, plus Dr. Steinman's initial report. He first attempted to rebut Dr. Steinman's arguments that Taxol-caused neuropathies would not typically present as motor neuropathies. *See generally* Second Callaghan Rep. at 1–2. Dr. Callaghan allowed that more severe forms of motor neuropathies caused by Taxol were uncommon, but stressed that *some* kind of motor neuropathy was still possible—and where observed, often involved leg weakness as a "prominent symptom." *Id.* at 1; H. Mo et al., *Association of Taxane Type with Patient-Reported Chemotherapy-Induced Peripheral Neuropathy Among Patients with Breast Cancer,* 5 JAMA Network Open 11:e2239788 (2022), filed as Ex. E Tab 1 (ECF No. 51-2) (Chinese prospective cohort study of approximately 1200 subjects, 41.7 percent of whom received Taxol; approximately 47 percent reported motor symptoms). He also noted that Petitioner had been deemed to suffer from a "grade three" motor neuropathy, consistent with Petitioner's presentation (and the grade designation did not require her to be non-ambulatory, as Dr. Steinman argued). Second Callaghan Rep. at 1.

[8] In support of this argument, Dr. Callaghan asserted that this onset to nadir timeframe is "required" by the Table. First Callaghan Rep. at 3. This contention is misleading. The Table only requires initial *onset* to occur in a defined timeframe. 42 C.F.R. § 100.3(a)(XIV)(D). However, the qualifications and aids to interpretation for GBS do indicate that the disease will usually have a nadir timeframe from onset consistent with Dr. Callaghan's argument—and therefore it is more than fair to assess, under the facts of this case, if nadir occurred as would be expected—and if not, why.

Other factors also made Taxol more likely the cause of Petitioner's total presentation, in Dr. Callaghan's view. Contrary to Dr. Steinman's emphasis on Petitioner's ongoing neurologic issues even in the absence of the Taxol treatment (suggesting no association), Dr. Callaghan pointed out that Petitioner had improved once the Taxol treatment fully ended (and without the usual lingering GBS sequelae)—albeit the improvement was witnessed long after, in the fall of 2013. Second Callaghan Rep. at 1 (*citing* Ex. 7 at 3). He also maintained that Dr. Steinman incorrectly contended that cessation of Taxol should result in immediate symptoms improvement, since "[c]hemotherapy-induced neuropathy is well-known to persist in some patients." Second Callaghan Rep. at 2; G. Cavaletti et al., *Chemotherapy-Induced Neuropathy,* 13 Current Treatment Options in Neurology 180, filed as Ex. E Tab 2 (ECF No. 51-3) ("Cavaletti"). Cavaletti is a review article, and it does confirm the possibility of progression or worsening of symptoms even after chemotherapy treatments cease (Cavaletti at 188), but (and although it does link motor symptoms with Taxol) does not provide harder data useful in assessing when this is more or less likely to occur.

Dr. Callaghan took issue with Dr. Steinman's arguments about other factors relevant to the proper characterization of Petitioner's injury. Relying on the conclusion that Petitioner's initial, pre-vaccination symptoms were both neurologic and not simply reflective of a distinguishable case of limited, Taxol-related symptoms, Dr. Callaghan maintained that her onset occurred during the first week of November 2012—meaning nadir (reached after her January 2013 hospitalization) took far too long for her condition to be deemed GBS. Second Callaghan Rep. at 2. In addition, he again pointed out that IVIG has not assisted her, further making a Taxol-associated neuropathy more likely. *Id.* Ultimately, Dr. Callaghan opined that Dr. Steinman was simply ignoring obvious record proof that Taxol was to blame for Petitioner's condition. Second Callaghan Rep. at 2.

Next, Dr. Callaghan responded to some of Dr. Schwartz's points about the Taxol-neuropathy association. Second Callaghan Rep. at 2–3. Although Dr. Schwartz had stressed that his own personal treatment experience had never involved a person with as severe a neuropathy as Ms. Rushing, Dr. Callaghan deemed the situation unsurprising, since the overall number of patients who would face similar debilitating symptoms was low. This did not mean, however, that it was unheard-of, and he referenced literature in support. *Id.* at 2; Jones at 5548.

Dr. Callaghan also disagreed with Dr. Schwartz's argument that Taxol dosage levels made a severe neuropathic response less likely. He noted that Freilich had observed neuropathies at doses of 200 mg/m^2, and becomes "dose limiting" (meaning treaters more often than not opt to reduce dosage in light of observable worsening of neuropathic symptoms in response to Taxol) at 250 mg/m^2—far less than the 960 mg/m^2 cumulative dose Petitioner had received, and thus more than enough to result in a more severe neuropathy. Freilich at 116. In Jones, moreover, "the median number of Taxol cycles" for the 200-plus evaluated patients was four, with 173 mg/m^2 per cycle—meaning such individuals also received lower cumulative doses than Petitioner, even though some

developed severe motor neuropathies as well. Second Callaghan Rep. at 3. Jones itself does not, however, correlate the severity of adverse outcomes to dosage specifically. Jones at 5548, Table 5.

Finally, Dr. Callaghan provided his reaction to Dr. Picot's second statement. Dr. Picot had asserted that he had "ruled out" a Taxol-caused neuropathy based on his examination of Petitioner, but Dr. Callaghan questioned whether any "examination feature" from the filed record supported that conclusion. Second Callaghan Rep. at 3. He again maintained that EMG findings could support a Taxol-induced neuropathy as much as GBS axonal neuropathy, and therefore they could not be reasonably relied upon to distinguish the cause. *Id.*

2. Dr. Kenneth McClain – Dr. McClain is a pediatric oncologist, and he provided two written reports. Report, dated June 10, 2021, filed as Ex. C (ECF No. 34-1) ("First McClain Rep."); Report, dated December 22, 2022, filed as Ex. F (ECF No. 51-5) ("Second McClain Rep."). Dr. McClain concluded that Petitioner's Taxol treatments best explained her neuropathic symptoms.

Dr. McClain attended the dual M.D./Ph.D. program at the University of Chicago School of Medicine from 1963 to 1973. *Curriculum Vitae*, filed as Ex. D (ECF No. 34-7) ("McClain CV") at 1. Thereafter, he completed a pediatric residency at The Johns Hopkins Hospital in 1976, postdoctoral training at the National Institutes of Health in 1979, and a Hematology/Oncology Fellowship at the Department of Pediatrics, University of Minnesota in 1981. *Id.* Dr. McClain is currently a Professor of Pediatrics in the Department of Pediatrics at Baylor College of Medicine. *Id.* at 3. He is board certified by the American Board of Pediatrics in General Pediatrics and Hematology-Oncology. *Id.* at 5. Dr. McClain has published approximately 200 articles, many regarding non-Hodgkin's and Hodgkin's lymphoma. *Id.* at 13–32. Dr. McClain reports that he has over 40 years of experience treating children with a variety of chemotherapy drugs that can cause neuropath, and that for the past 25 years, he as also treated and cared for adult patients with histiocytic diseases. First McClain Rep. at 1.

First Report

Dr. McClain's first report included a short summary of Petitioner's disease course timeline, before getting to the meat of his opinion. First McClain Rep. at 1–3. He stressed from that history the fact that Petitioner's initial symptoms had begun as early as mid-November 2012 and were "consistent with grade 3-4 Taxol toxicity," given their sensory character. *Id.* at 3. But the literature on Taxol-induced neuropathies was also consistent with it causing more serious kinds of neuropathy—and thus "there is clear precedent for ascribing the [P]etitioner's motor neuropathy to Taxol." First McClain Rep. at 3.

The likelihood that a flu vaccine, by contrast, had been causal of Petitioner's GBS was low, in Dr. McClain's opinion. Given that literature established that flu vaccine-associated GBS was already rare—and that the axonal variant was one of the "least frequent" versions, the probability that the flu vaccine in this case had caused her GBS was especially unlikely. In comparison, studies like Jones revealed that a larger percentage of breast cancer patients receiving Taxol treatments experienced "neuromotor deficits." Jones at 5548, Table 5.

Second Report

The next written report offered by Dr. McClain repeated some of his prior review of the record, although in so doing he emphasized points from the record in which treaters had squarely attributed Petitioner's symptoms to her Taxol treatments. Second McClain Rep. at 2. He also observed that the two treaters offering opinions in this case (Drs. Schwartz and Picot) had noted the extent to which Petitioner's exam findings, symptoms, and testing results were not reflective of "classic GBS," but instead something atypical (even if they also discounted the Taxol therapy as causal). *Id.* And other treaters (including Dr. Schwartz himself) had not firmly embraced the flu vaccine as causal. *Id.* at 2–3.

Dr. McClain then took aim at the contention of the experts acting on Petitioner's behalf, all of whom maintained Taxol was not the cause of her overall neuropathic presentation. Even if it were true, for example, that most Taxol patients would only experience sensory-style symptoms, there are always patients who are outliers from the usual extent of drug side effects," and thus (since in rare cases more severe neuropathies *had* been associated with Taxol), the same could be the case here. Second McClain Rep. at 3. Petitioner's own circumstances (in particular, her weight loss) likely increased her vulnerability to a severe side-effect. *Id.*

Also significant to Dr. McClain was the nature of Taxol. Like Dr. Steinman, he observed that its effectiveness in treating cancer (and specifically the killing of breast cancer cells) was a function of its impact on the cellular microtubule system—which had the secondary capacity to damage nerve cells. Second McClain Rep. at 3. Aware of this side-effect, medical researchers had investigated genetic polymorphisms that might explain why some individuals would experience Taxol-associate neuropathic symptoms., identifying one genetic variant in particular as so associated. S. Park et al., *Paclitaxel-Induced Neuropathy: Potential Association of MAPT and GSK3B Genotypes,* 14 BMC Cancer 993 (2014), filed as Ex. F Tab 2 (ECF No. 51-7) ("Park") at 996 (study of 21 Taxol-treated patients (most of whom had breast cancer) determined (based on prevalence of specific genetic testing results) that "patient-specific factors such as genetic polymorphisms may be important in identification of at-risk patients"). In Dr. McClain's view, the risk could be multiplied by other genetic factors as well—thereby increasing "overall neurotoxicity risk." Second McClain Rep. at 3 (quoting Park at 995). In this case, however, there is no identified

genetic testing results that might make more concrete the findings of Park to Ms. Rushing's experience.

Another study had identified even more gene variants that, if present, were likely to compound the risk of a Taxol-induced neuropathy. G. Boora et al., *Testing of Candidate Single Nucleotide Variants Associated with Paclitaxel Neuropathy in the Trial NCCTG N08CI (Alliance)*, 5 Cancer Medicine 4:631 (2016), filed as Ex. F Tab 4 (ECF No. 51-9) (genetic testing aimed at identifying certain genetic variants more commonly associated with Taxol-induced neuropathy). Petitioner's reaction was likely attributable to some genetic-derived susceptibility—although Dr. McClain did not identify whether Petitioner did likely possess this susceptibility (beyond noting her clinical experience), or that axonal neuropathies post-Taxol *only* occurred under such circumstances.

III. Procedural History

After the case's initiation in March 2019, this matter was originally assigned to SPU, since it appeared to assert a flu-GBS Table claim (and thus potentially could be resolved quickly if the claim met the Table elements). Petitioner filed medical records supporting the claim, and then Respondent's Rule 4(c) Report was filed on May 22, 2020 (ECF No. 22). In the midst of the filing of the expert reports and treater statements discussed above, I had set the matter for an entitlement hearing, based on the nature of the dispute regarding the chemotherapy treatment as an alternative cause. *See* Prehearing Order, dated August 16, 2022 (ECF No. 40). However, the parties later informed me of their preference for the claim to be resolved on the papers, and I accordingly set a schedule for briefing their positions. Order, dated September 6, 2022 (ECF No. 47). After some schedule modifications, briefing was completed, and the matter has been ripe for decision since August 2023.

IV. Parties' Arguments

Petitioner argues that she has met her *prima facie* burden of demonstrating a Table Injury[9]—she received a flu vaccine, experienced GBS, and her onset fell within three to 42 days of vaccination. Br. at 21. The only remaining issue was whether Respondent had met his burden of establishing a factor unrelated to the flu vaccine—something Petitioner denied had occurred. *Id.*

Petitioner maintains that her case "is unique in that her treating oncologist and neuromuscular specialist both authored a total of five reports supporting her claim and clarifying that, based on their first-hand knowledge of her condition and their in-person physical exams of

[9] Petitioner also maintains he could meet all three *Althen* prongs for a non-Table case. Opp. at 23–24, 26.

her, they ruled out Taxol neuropathy as a cause of her symptoms." *Id.*; Exs. 15, 16, 43, 60, 70. Moreover, none of Petitioner's treating physicians attributed her lower extremity paralysis to her Taxol therapy during her hospitalization. Mot. at 21–22. Petitioner further notes that the neurotoxicity of Taxol is dose-dependent, and that Respondent's experts were unable to offer a study where a patient developed a grade four motor neuropathy from a dose as low as hers. *Id.* at 24.

Respondent argues that Petitioner's neuropathic symptoms were attributable to the Taxol chemotherapy regime she had unquestionably undergone pre-vaccination. Respondent notes that Taxol "can, and likely did," cause Petitioner's neurologic symptoms—further noting that "Taxol is a well-known cause of sensory neuropathy occurring in as many as 49-67% of patients." Opp. at 18; First Callaghan Rep. at 4 (citing Freilich at 115). Moreover, Respondent maintains that "the risk of developing GBS after an influenza vaccination is less than one in three million." Opp. at 18; First Callaghan Rep. at 4. Thus, the likelihood of developing the form of GBS experienced by Peitioner—axonal GBS—is even lower as "axonal GBS only accounts of 3-17% of all cases in North America. First Callaghan Rep. at 4; Kuwabara at 1181. Respondent contends that the medical records do not support a diagnosis of axonal GBS, but instead "the evidence supporting Taxol neuropathy is clear with [Petitioner's] clinical history, examination, and timing of symptoms starting during treatment." Opp. at 19.

V. Applicable Legal Standards

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[10] In this case, Petitioner asserts the Table claim of GBS caused by the flu vaccine.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's

[10] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

B. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs.*, 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to

conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs.*, No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs.*, 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs.*, No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the

result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743

(quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs.*, No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

E. *Standards for Ruling on the Record*

I am resolving Petitioner's claim on the filed record, based on the parties' decision to forego the hearing I had originally scheduled. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs.*, No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs.*, 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

ANALYSIS

I. Petitioner Has Met Her Prima Facie Burden of Proof for a Table Flu Vaccine-GBS Claim

GBS is listed as a Table injury for the flu vaccine, and thus a claimant seeking to meet its requirements need not establish vaccine causation. Instead, Petitioner herein must show (a) receipt of a covered form of the flu vaccine, (b) that she did in fact experience GBS, as defined in the Table's "qualifications and aids to interpretation," and (c) that her symptoms onset (whether or not GBS could then be diagnosed, or was) occurred between three and 42 days after vaccination. 42 C.F.R. § 100.3. There is no dispute that Petitioner received a version of the flu vaccine covered by the Program, but some disagreement as to whether (a) Petitioner actually experienced GBS, (b) its onset occurred in the timeframe set for the claim, and/or (c) Petitioner's neuropathy was "drug-induced"—caused by her undisputed Taxol treatments. Although these matters were reasonably disputed, and resolution of some of the items present close calls, I determine that the balance of evidence favors Petitioner.

First, the medical record supports Petitioner's favored diagnosis of an axonal form of GBS. The Table itself recognizes that *some* GBS variants are cognizable claims, and this includes the axonal form, often referred to with the acronym "AMAN" (or "acute motor axonal neuropathy").[11] *Swaiss v. Sec'y of Health & Hum. Servs.*, No. 15-286V, 2019 WL 6520791 (Fed. Cl. Spec. Mstr. Nov. 4, 2019). Thus, the fact that Petitioner's form of GBS is less common does not render the diagnosis suspect, assuming it has record support. As it stands, Petitioner's favored diagnosis *does* have such support. Ex. 6 at 5, 12, 67. And Petitioner has also offered the opinion of a contemporaneous treater who actually saw Petitioner, Dr. Picot (though I give far more weight to his second statement than his first, conclusory comment). First Picot Statement at 1; Second Picot Statement at 1. Dr. Callaghan's alternative reading of the record is reasonable, and he does observe findings not fully consistent with the proposed GBS diagnosis. But although there are cases where the record is sufficiently ambiguous to cause me to look to the opinions of after-the-fact experts for guidance or interpretation, giving them greater weight, here the record itself is enough to support Petitioner's contention on diagnosis.

Second, I find that it is not preponderantly likely that Petitioner's Taxol treatments were causal of her GBS—even though the record *does* establish she had the kind of common neuropathic symptoms associated with Taxol while, and not long after, she was receiving it as chemotherapy. It is undisputed that Taxol can cause *some kinds* of neuropathy—more often than not a mild, limited sensory form, but also potentially a motor/axonal form. But Petitioner's expert group persuasively established that the relatively low dosage levels Petitioner received are not usually

[11] By contrast, a different variant—chronic immune demyelinating polyneuropathy, or "CIDP"—is a defined exclusionary diagnosis that, if present, means a Table flu-GBS claim cannot be substantiated. Section 14(b).

associated with severe neuropathies. Petitioner's efforts to undermine the contention that Taxol explained her symptoms gained significant help from Dr. Schwartz—an oncologist with direct experience treating Petitioner, unlike Dr. McClain. Dr. Schwartz in fact represented that, in his experience, he had never seen one of his patients develop such a severe post-chemotherapy neuropathy—an assertion I give greater weight than Respondent's objection that it remained "possible" Taxol could also in rare cases result in a worsened form of neuropathy. I would require more evidence than such reasoned speculation to find it preponderantly the case.

Many filed items of literature confirm Petitioner's assertions about the association between Taxol dosage and degree of likely neuropathy. Jones at 5548; Freilich at 117–18; Scripture at 166, 167. By contrast, no literature goes so far to identify GBS *itself* as a Taxol-associated injury, or that in most cases a severe form is likely at the dosage levels Petitioner experienced. And in many regards, the record itself is more consistent with Petitioner having experienced *two* forms of neuropathy rather than one continuous and progressive condition. Ex. 6 at 9. Her worsening also occurred well after the Taxol treatments had ended—something else rendering the facts inconsistent with those studies, although I acknowledge Respondent's point that Taxol-caused neuropathies can progress even once treatment ends.

No doubt it has been established on this record that Taxol *can* be associated with more severe symptoms as well, approaching even what Petitioner experienced. But Petitioner's experts persuasively explained why that was not likely *under the facts of this case*, offering credible literature in support. At bottom, the dosage level Petitioner received was not likely high enough to put her into the category of subjects from the studies discussed in Scripture.

Respondent's experts' arguments to the contrary—that it is *possible* Taxol could cause GBS-like symptoms, and that this was statistically more likely than the flu vaccine—have some facial appeal. It was certainly *reasonable* for Respondent to take note of the Taxol chemotherapy regime, and question its causal role. It is undisputed Petitioner received it and experienced some symptoms attributable to it—something even Dr. Schwartz felt had occurred (although he later felt her degree of worsening was inconsistent with that as the cause). Taxol is definitely associated with neuropathic symptoms. Indeed, some of the literature offered in this case reflects efforts by medical science to evaluate how, or when, to administer Taxol, given its understood side-effects. *See, e.g.*, Timmins at 367, 68–69. I also deem relevant and worthy of consideration the articles filed that suggest Taxol-induced neuropathies may have a genetic-susceptibility component that explains why all Taxol-receiving patients do not go on to develop neuropathies—although there was no evidence in this case that Petitioner was tested genetically or possessed the putatively-associated variants (and their causal role was unquestionably not shown, in any event, to exceed the role Taxol would play).

Ultimately, Respondent to no small extent seemed to rely on the argument that Taxol can *possibly* and in rare circumstances cause severe neuropathies, while emphasizing a generally low statistical likelihood that the flu vaccine can cause GBS. Of course, the second argument flies in the face of the claim's Table nature (and could only be overcome by showing of "factor unrelated"—which Respondent did not successfully achieve).[12] But otherwise, these are the kind of arguments I often *reject* when a petitioner puts them forward in favor of causation. The mere *possibility*, no matter how well reasoned, of something happening is not the same as a determination it is "more likely than not." In this case, the record persuasively establishes that Taxol can likely cause *some* neuropathic symptoms, including motor-related—and that Petitioner's initial sensory symptoms were likely due to her chemotherapy regime. But the argument that vaccine injuries are more rare than Taxol neuropathies does not undermine the conclusion that *in this case* the Taxol was not the likely cause of Petitioner's symptoms—any more than the argument oft-advanced by Program claimants that the rarity of vaccine injuries mean their burden of proof must be adjusted downward. Thus, although this record establishes some initial, transient sensory neuropathic symptoms due to Taxol, it does not also establish that the second "round" of symptoms flowed from the first.

Finally, the record preponderates in favor of the determination that Petitioner's onset of GBS-specific symptoms (which the record shows can be distinguished from her Taxol-associated sensory neuropathic symptoms) occurred within the onset timeframe set by the Table—although this issue presents the closest fact dispute between the parties. I decide it in Petitioner's favor—but just barely.

There is some ambiguity in the record about when Petitioner's symptomatic issues transitioned, from milder sensory issues to the more severe motor concerns she later expressed.

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[12] On the basis of this same record and relevant evidence, I could not find that Respondent carried his "factor unrelated" burden (based on my initial determination that Petitioner met her prima facie burden of proof). *Stone v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 233, 237 (2010). One important aspect of that burden is the obligation placed on Respondent to *rule out* vaccine causation—something A Petitioner need not do when establishing her prima facie case. *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1351, 1354 (Fed. Cir. 2008).

In essence, although it is clearly established that Taxol can *sometimes* cause severe neuropathic symptoms affecting motor nerves, I do not find the flu vaccine's role can on this record be completely eliminated as substantial—especially since Petitioner's experts persuasively demonstrated that the dosage levels at issue never became high enough to likely be so damaging. Thus, even if this were a case (and it could well be) that the *interaction* between Taxol and the impact of the flu vaccine caused Petitioner's later GBS, that finding would not eliminate the vaccine as causal in some substantial part—and hence Respondent's somewhat more difficult burden on the issue of "factor unrelated" was not met. *Id.* There are cases where this showing can be met. *See, e.g.*, *White v. Sec'y of Health & Hum. Servs.*, No. 20-1319V, 2023 WL 4204568, at *17 (Fed. Cl. Spec. Mstr. June 2, 2023), *mot. for review den'd*, 168 Fed. Cl. 660 (2023), *appeal docketed* No. 2024-1372 (Fed. Cir. Jan. 23, 2024) (infection Petitioner possessed was far more likely to cause GBS than the flu vaccine). Here, Taxol has been shown by Petitioner *not* to be commonly associated with the axonal form of GBS—and to be uncommonly causal of axonal neuropathies more generally. I thus do not give much weight to Respondent's expert's efforts to elucidate the comparative statistical commonality of flu vaccine-caused GBS versus Taxol neuropathies.

But the record does establish that her complaints became qualitatively different in the second half of December, toward the end of the month, when the numbness and tingling in her legs, feet, and hands persisted, causing Petitioner to suffer multiple falls due to continued weakness. Ex. 13 at 34–39. Thereafter, it took treaters some time to coalesce around a GBS diagnosis—but this consensus is not the same as when symptoms first *manifested,* which is of course the Program's only concern when attempting to determine whether onset occurred in a Table-designated timeframe. *Nieves v. Sec'y of Health & Hum. Servs.*, No. 18-1602V, 2023 WL 3580148, at *40 (Fed. Cl. Spec. Mstr. May 22, 2023), *mot. for review den'd*, 167 Fed. Cl. 422 (2023). Here, the Table provides for up to 42 days for onset—and thus any onset by the last week of December would fall into that timeframe. Dr. Callaghan's nadir arguments do not undermine this conclusion—for measuring from this onset (rather than when Petitioner first experienced the distinguishable, Taxol-associated symptoms), it can be concluded that Petitioner's condition progressed downward over the ensuing four weeks, causing her to "hit bottom" by "1/25/13." Ex. A at 4; 42 C.F.R. § 100.3(c)(15)(ii)(C).

While these points find sufficient record and expert support for me to be comfortable to conclude as I do, there is no doubt that *overall* this record is flush with factual ambiguities. Taxol's association with neuropathy as a side-effect is fully-established. Taxol treatments can also, less commonly, result in more severe motor neuropathies. And those symptoms do not vanish simply when treatment ends, as Petitioner struggled to establish. It was reasonable to suspect Petitioner's initial symptoms were the start of what she later experienced—indeed, treaters who saw her in December 2012 and even closely thereafter thought this to be the case, at least initially (even if they rationally altered their views later on, after added treatment experience with her). And not only did Petitioner experience an uncommon form of GBS, but the diagnosis itself is not fully confirmed by the record.

But the coin of the realm in the Vaccine Program, evidentiarily-speaking, is *preponderance,* not certainty—and close cases can tip in a petitioner's favor even when other credible evidence suggests an alternative conclusion. In fact, case law encourages special masters to decide such evenly-disputed matters *for* claimants. *Strong v. Sec'y of Health & Hum. Servs.*, No. 15-1108V, 2018 WL 1125666, at *20 (Fed. Cl. Spec. Mstr. Jan. 12, 2018). Just as there are many cases where I find a petitioner has not crossed the preponderant "line," even though some reliable evidence might favor his claim, the opposite can be the case as well—and is so here.

II. The Claim is Timely Under the Act's Lookback provision

In many prior determinations, I have ruled that only "new" Table-version claims involving a vaccine previously added to the Vaccine Program are "saved" by the Act's lookback provision. *Randolph v. Sec'y of Health & Hum. Servs.*, No. 18-1231V, 2020 WL 542735, at *9 (Fed. Cl.

Spec. Mstr. Jan. 2, 2020).[13] Here, Petitioner's claim is facially untimely, and thus can only go forward if the lookback's operations protect it. I find they do. As noted, the claim alleges a successful Table claim of GBS after the flu vaccine. The matter was filed within two years of amendment, and eight years from date of injury (since less than seven years passed from Petitioner's likely onset in mid to late-December 2012 and the filing of the claim in March 2019). Thus, because I have found the Petition advances a meritorious Table claim, it is saved by the lookback provision.

CONCLUSION

Based on the entire record in this case, I find that Petitioner has provided preponderant evidence satisfying all requirements for a Table-GBS claim. Petitioner is thus entitled to compensation. An order establishing a schedule for resolution of damages shall follow.

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[13] Other special masters have taken a more expansive view of the lookback provision, and found it also saves certain causation claims, reasoning (in a "rising tide lifting all boats" manner) that the mere existence of a new Table claim makes comparable, but non-Table, causation claims more likely to succeed as well. *See e.g.*, *Simpson v. Sec'y of Health & Hum. Servs.*, No. 17-944V, 2019 WL 11815360 (Fed. Cl. Spec. Mstr. Aug. 7, 2019) (46 to 47-day onset of GBS after flu vaccine too long to constitute Table claim, but non-Table claim deemed timely filed based on lookback provision). I of course am not bound by the decisions of other special masters, but I deem this reasoning unpersuasive. It blurs the recognized distinction between Table claims (where the Government concedes causation if certain facts are established) and causation claims. It also ignores the fact that there is a difference between "table" amendments that allow new vaccines to be the basis of a claim, versus amendments adding a designated new injury. The ability to claim a flu vaccine-related injury greatly predated the Table amendment adding the flu vaccine-GBS claim.